



PATENT

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Client Reference UC 80-065-4

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(Marian Christopher)

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In the application of:

MILLER, et al

Serial No.: 08/487,312

Filing Date: June 7, 1995

For: BOVINE GROWTH HORMONE

Examiner: C. Saoud

Group Art Unit: 1646

**Declaration of Robert B. Petersen, Ph.D.
Pursuant to 37 C.F.R. § 1.132**

MS Non-Fee Amendment
Commissioner for Patents
P.O. Box 1450
Alexandria, VA 22313-1450

Dear Sir:

I, Robert B. Petersen, Ph.D. declare as follows:

1. I am an Associate Professor of Pathology and Neuroscience at Case Western Reserve University.
2. I have been conducting research in the field of prion disease for 12 years. My research expertise encompasses the fields of cell and molecular biology of prion disease. My education, awards and honors, professional society memberships, relevant training and work experience, publications,

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published abstracts and invited lectures and committee memberships are set forth in the attached Curriculum Vitae.

3. I have worked with recombinant models for the past 19 years in which proteins were expressed and analyzed.
4. The objective of this declaration is to evaluate the risks of pituitary derived versus recombinant derived bovine growth hormone for the development of Mad Cow disease.
5. Prior to preparing this declaration, I reviewed and became familiar with the following: (1) U.S. Patent No. 3,265,579 (hereinafter "Daniel's"); (2) U.S. Patent Application No. 487, 312 (hereinafter the "312 Application"); (3) the opinion of the U.S. Board of Patent Appeals and Interferences, Appeal No. 1998-1851 (March 26, 2003); and (4) Prusiner, Proc Natl Acad Sci USA **95**: 13363.
6. I have concluded that recombinant bovine growth hormone poses essentially no risk of causing Mad Cow disease whereas the pituitary derived hormone has a high risk, if Mad Cow disease is present in the population.

Bovine Growth Hormone

7. Bovine growth hormone, BGH, is a bovine derived protein comprised of 191 amino acids. BGH is produced in the pituitary, which is located deep in the brain near the brainstem. Since hormones are produced in the body in vanishingly small quantities, BGH is purified from the pooled pituitary glands from many cattle. These pools are typically on the order of greater than 1,000 pituitary glands.
8. A recombinant form of BGH is used in agriculture in the US to promote growth and milk production in cows. The recombinant BGH is administered to cows by subcutaneous injection

[<http://www.monsantodairy.com/about/benefits/att3.html>;
<http://www.fda.gov/cvm/efoi/section2/140872.pdf>]. The recombinant form is produced using DNA vectors transformed into unicellular organisms.

BSE

9. Bovine spongiform encephalopathy, BSE, or "Mad Cow" is a disease of cattle that was recognized in the United Kingdom in the mid-1980s. The disease was found to be a prion disease. Prions are a novel class of infectious agents lacking a nucleic acid genome and comprised primarily, if not only, of protein. The prion protein, which is a normal cellular protein, is believed to undergo a change in conformation rendering it pathogenic.
10. BSE is believed to have arisen from the ingestion of contaminated feed derived from scrapie-infected sheep. Scrapie was the first prion disease recognized and affects sheep and goats. Once cattle became infected, a cycle was formed due to the practice of using rendered cattle in feed. From cattle, the disease spread to the London zoo, domestic cats, and, in 1996, the first recognized case of the human disease variant Creutzfeldt-Jakob disease (vCJD).
11. While the UK, Europe and Japan currently test cattle for BSE only limited testing is done in the US. Testing is both difficult and expensive, taking at least a day to perform. These tests were not available prior to the late 1990s. In fact, the first case of BSE was not noted until around 1985. The tests currently in use use the brainstem as the test substrate since it contains uniformly high amounts of the prion. The gold standard test is the bioassay in which brain homogenate is injected into the brain of a susceptible animal. However, this test is not practical for routine screening since the latency period before disease development is in excess of 60 days.

12. BSE has the potential to severely impact the US economy as evidenced by the regulation of its possession, use or transfer (9CFR121.3) promulgated under the Public Health Security and Bioterrorism Preparedness and Response Act of 2002 (PL 107-188). In the years following the outbreak of BSE, the UK is estimated to have lost approximately \$3 billion to the ban of importation of beef from the UK. Due to the difference in size of the US cattle industry, the impact of BSE on the US would be greater by at least an order of magnitude.
13. Given the economic and safety risks BSE presents, regulatory agencies should not allow the use of pituitary derived BGH in cows, especially given the availability of the safe recombinant form.

CJD

14. Creutzfeldt-Jakob disease, CJD, is a progressive neurological disease of humans that occurs sporadically, by inheritance, or by infection. It is also believed to be caused by prions. Both CJD and vCJD are invariably fatal and diagnosis is only possible post-mortem.
15. Infection of humans with CJD has come about by the use of contaminated corneas and dura mater grafts, surgical instruments, growth hormone and contaminated beef. The last cause results in variant CJD, vCJD, that has been found in individuals decades younger than the age typically associated with CJD, 28 years old versus 55-60 years of age. The correlation of vCJD with BSE resulted in plummeting demand for beef in the UK and Europe. For example, after the discovery of BSE in Italy demand for beef dropped 70% overnight.
16. CJD has also been caused in humans who received human growth hormone derived from pools of human pituitaries. Human growth hormone has been used to treat some forms of growth disorders since the 1960s. In the 1970s in the US and the UK use of pituitary derived growth

hormone contaminated by prions resulted in CJD in the recipients. This unfortunate occurrence was repeated in France the mid-1980s where two contaminated lots of growth hormone were produced. Over 200 of the children treated with the contaminated growth hormone developed CJD.

17. These cases of iatrogenic CJD prompted the adoption in the US and Europe of using recombinant human growth hormone for treatment (rather than the previously used pituitary derived form) since recombinant production precludes the co-purification of the prion disease agent, and consequently has no risk of transmitting CJD. (This is discussed in more detail, below.)

BSE Risk Associated with Pituitary Derived BGH

18. There is a risk of BSE associated with the use in cattle of pituitary derived growth hormone.
19. Central nervous system tissue, including the brainstem, is a source of infectious prions that cause BSE.
20. The location of the pituitary gland makes it susceptible to contamination from infectious agents found in the brain and particularly from the brainstem. It is difficult to extract the pituitary from the brain without contamination. This is compounded by the pooling of thousands of pituitary glands to isolate useable amounts of growth hormone.
21. If pituitaries in the pool used to purify the pituitary derived form of BGH were infected, then standard purification techniques (like those described in Daniels) would not remove the BSE agent. (In other words, the Daniels method would co-purify the BSE agent if it was present in the pituitary starting material.)

22. Moreover, the method of administering cows with BGH (inoculation) would allow transmission of BSE if the hormone used was contaminated, as occurred in the human cases (see above).
23. The risk with pituitary derived BGH is enhanced by the fact that (a) testing cows for BSE, or testing samples of the BGH, is difficult and not routinely done in the US; (b) many pituitaries are needed to get enough hormone for use; (c) if a cow were infected, it would be difficult to extract pituitary without contamination from brain/brainstem resulting in contaminated BGH.

Recombinant BGH

24. The '312 Application describes the production of bovine growth hormone through recombinant technology. That is, it describes the production of the hormone by transforming an organism with the gene that encodes BGH and causing the organism to express relatively large quantities of the hormone.
25. The prion protein, which is the major if not only constituent of prions, has only been found in mammals.
26. Recombinant proteins are generally produced by heterologous expression in bacteria. The switch to a bacterially produced recombinant hormone would prevent the possibility of co-purifying a prion since the bacteria is incapable of making a prion.
27. In addition, the reagents used in the production of the recombinant hormone are also without risk for providing contaminating prions making the purified recombinant growth hormone essentially risk free.

28. The use of a recombinant protein source results in an easier purification and a product of higher purity than the form of hormone purified from pituitary tissue. The ease of purification is the result of all cells in the sample expressing high levels of the recombinant protein whereas in the tissue only some cells express and that at low levels.
29. Additionally, the production of the BGH by recombinant methods yields only one isoform of BGH. In comparison, natural BGH is produced as four different allelic and splice variants yielding four BGH isoforms. In short, pituitary derived BGH will consist of multiple isoforms whereas the recombinant BGH will consist of only one isoform.

Declaration

30. I hereby declare that all statements made herein of my own knowledge are true and all statements made on information and belief are believed to be true and further these statements were made with the knowledge that willful false statements and the like are punishable by fine or imprisonment or both under Section 1001 of Title 18 of the United States Code, and such willful false statements may jeopardize the validity of the application, any patent issuing thereon, or any patent to which this verified statement is directed.

Date: 3/24/04

Signed: 

Robert B. Petersen, Ph.D.

Robert B. Petersen

March 26, 2004

CURRICULUM VITAE

**Robert B. Petersen, Ph.D.
Associate Professor**

Personal Information:

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Fax:360-838-9226; e-mail: rbp@po.cwru.edu
Home Address: 32280 Creekside Drive
Pepper Pike, Ohio 44124
216-292-3589

Education:

1981 BS, Biochemistry, University of Minnesota, Graduated Magna Cum Laude

1985 Ph.D., Cell and Developmental Biology, University of Minnesota, NIH pre-doctoral trainee on Genetics training grant; American Cancer Society Institutional Grant recipient. Department of Genetics and Cell Biology, Advisor: Dr. Perry Hackett, 9/81 - 7/85

1986 Postdoctoral Research Fellow, University of Minnesota, Human Genetics Institute, Advisor: Dr. Perry Hackett, 8/85 - 10/86

1990 Postdoctoral Research Fellow, The University of Chicago, Awarded 3 year NIH National Research Service Award. The University of Chicago, Department of Molecular Genetics and Cell Biology, Advisor: Dr. Susan Lindquist, 11/86 - 1/90.

Faculty Appointment:

Instructor, The University of Chicago, Department of Molecular Genetics and Cell Biology, 2/90 - 8/91.

Assistant Professor, Department of Pathology, Case Western Reserve University, Cleveland, Ohio, 9/91 - 6/98.

Associate Professor, Department of Pathology, Case Western Reserve University, Cleveland, Ohio, 7/98 - present

Teaching Experience:

Robert B. Petersen

1992-98 Phase II, Nervous system, lecturer
1993 Path 516 - Experimental Pathology, lecturer
1997- Path 444- Neurological Diseases: Pathological, Cellular, and
 Molecular Perspectives. Co-organized with MA Smith and
 lecturer
2002- Anat 523, lecturer

Students advised:

1992 Tracy Craig, summer undergraduate research program
1992 Chris Erhardt, medical student
1992-93 David Shein, pathology fellow (co-sponsored with Dr. Mark Cohen)
1993-95, 97-99 Ishrat Zaidi, post-doctoral student
1996 Lorie Raetzman, rotating graduate student
1995-1998 Sabina Capellari, visiting resident
1997 Jeremy Sanford, rotating graduate student
1998-1999 Amy C. Long, graduate student

Residents advised

1996 Nancy Tresser, M.D., neuropathology fellow
1995 Rudolph Castellani, M.D., neuropathology fellow

Thesis Advisory Committee:

1996 Tobin Cheung, Pathology (S.G. Younkin advisor)
1998 Dacai Liu , Pathology (M. Sy advisor)
1998 Rosa Rivera, Biochemistry (outside reviewer)
1998 Xiang-Hong Song, Pathology (J. Teller advisor)
1998- Krista Seanor, Pathology (D. Templeton advisor)
1999- Tao Tao. Pathology (A. Tartakoff advisor)
1999- Tong Liu, Pathology (M. Sy advisor)

Committee chair for Dacai Liu and Xiang-Hong Song

Research Support (direct costs):

Active

Co-PI of entire project. National Institute of Health, Program Project, AG14359: Pathogenetic Mechanism of Prion Disease. \$2,962,578 (6/97 - 5/02).

Principal Investigator Project 1(Program project AG14539): Transgenic models of prion diseases linked to the D178N mutation. \$200,993 (6/99 - 5/00).

Past:

Co-Investigator. National Institute of Health, AG08155, Method to Extend Research in Time Award (MERIT), 04/01/94 - 03/31/99 (Pierluigi Gambetti, Primary applicant). \$1,140,185.

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Principal Investigator Project 3, Heat shock system: A model relevant to Alzheimer disease. National Institute of Health, Leadership and Excellence in Alzheimer Disease (LEAD) Award, 07/01/91 - 06/30/98 (Pierluigi Gambetti, Primary applicant). \$464,181

Principal Investigator Pilot Project, Cellular and Molecular Pathology of Alzheimer Disease. National Institute of Health, Leadership and Excellence in Alzheimer Disease (LEAD) Award, 07/01/91 - 06/30/98 (Pierluigi Gambetti, Primary applicant). \$45,557

Ad hoc Journal reviewer:

American Journal of Human Genetics

Ad hoc Journal reviewer (continued):

American Journal of Pathology

Biochemistry

BioTechniques

Brain Pathology

Journal of Biological Chemistry

The Lancet

Neurology

Journal of Neuroscience

Clinical Genetics

EMBO

Ad hoc Grant Reviewer:

Nebraska Experimental Program to Stimulate Competitive Research

1998 United States Department of Agriculture; Animal Health and Well-Being Program

1999 National Institute of Health, National Heart, Lung, and Blood Institute

Special Emphasis Panel, Development of Assay Methods for CJ Disease, RFA-NIH-HL-99-003.

2001 United States Department of Agriculture; Animal Genome

Professional Societies:

American Association for the Advancement of Science, 1981- present

American Society for Microbiology, 1984-present

American Society for Neuroscience, 1992- present

American Chemical Society, 1995-199

American Society for Cell Biology, 1997- present

Committees:

Department

Pathology Seminar Committee, 1993- present

Pathology Seminar Committee, Chair and Co-Director, 1994-98

Pathology representative to the BSTP Admissions Committee

School of Medicine

Robert B. Petersen

Member of the Alzheimer Center 1992- present
Judge: Irwin Lepow Medical Student Research Day, 1995.

Case Western Reserve University

Institutional Animal Care and Use Committee (IACUC)-1997- present
Institutional Animal Care and Use Committee (IACUC)- Chair 1998-2001.
Institutional Animal Care and Use Committee (IACUC)- Executive Committee- 1997-2001.
Judge: Michaelson - Morley Undergraduate Research, 1994, 1995.

Conference Committees:

Scientific Advisory Committee: Neurodegenerative Disorders: Common Molecular Mechanisms. Ocho Rios, Jamaica. February 23-28, 1997.

Scientific Advisory Committee: Neurodegenerative Disorders: Common Molecular Mechanisms. Ocho Rios, Jamaica. February 23-28, 1998.

Session Chair

Neurodegenerative Disorders: Common Molecular Mechanisms. Molecular Biology and Genetics. Ocho Rios, Jamaica. April 10-15, 1994.

Neurodegenerative Disorders: Common Molecular Mechanisms. Neuropathology: Clinical and Experimental Aspects. Ocho Rios, Jamaica. April 2-7, 1995.

Neurodegenerative Disorders: Common Molecular Mechanisms. Spongiform Encephalopathies: Experimental. Ocho Rios, Jamaica. February 23-28, 1997.

Neurodegenerative Disorders: Common Molecular Mechanisms. Prion Diseases. Ocho Rios, Jamaica. February 23-28, 1998.

Neurodegenerative Disorders: Common Molecular Mechanisms. Infectious Agents. Tobago, West Indies. April 8-14, 2000.

Consulting

Scientific Consultant- Efoora, Inc. (1/99-8/2000)
Chief Scientific Officer- Prion Developmental Laboratories, Inc. (9/2000-
Scientific Advisor, Genesis Bioventures, Inc. (4/01-11/02)
Director- Prion Developmental Laboratories, Inc. (1/2003-

Invited Seminars/ Platform Presentations:

1. Petersen RB, McGarry TJ, DeBenedetti A, Golic KG, Lindquist S. Translational control during the heat shock response in *Drosophila*. *ASBC Fed Proc* 46: 1958, 1987.

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2. Petersen RB, McGarry TJ, Golic KG, Dellavalle RP, Lindquist S. Translational control during the heat shock response of *Drosophila*. *Regulation of Gene Expression by RNA Structure and Anti-messengers*, NATO/INSERM Workshop, Les Arcs, Savoie, France, February 1988.
3. Petersen RB and Lindquist S. Regulation of the *Drosophila* Hsp 70 gene. *Translation Control*, Cold Springs Harbor, September 1989.
4. Petersen RB, Vogel J, McGarry TJ, Lindquist S. Post-transcriptional regulation of the heat shock response in *Drosophila*. *Post-Transcriptional Control of Gene Expression*. NATO/EEC Workshop, Goslar, W. Germany, 1990.
5. Petersen RB, Goldfarb L, Tabaton M, Brown P, LeBlanc A, Montagna P, Cortelli P, Monari L, Autilio-Gambetti L, Gajdusek DC, Lugaresi E, Gambetti P. Fatal familial insomnia and one subtype of familial Creutzfeldt-Jakob disease: Effect of a polymorphism in the prion protein gene on a pathogenic mutation. *Neurology*, Vol 43, no. 4. *Am Acad Neurol 45th Annual Meeting*, April 1993.
6. Petersen RB, Tabaton M, LeBlanc A, Montagna P, Cortelli P, Monari L, Autilio-Gambetti L, Lugaresi E, Gambetti P. Fatal familial insomnia and the expanding family of prion diseases. *Transmissible and Non-Transmissible Neurodegeneration Disorders: Current Update*, Ocho Rios, Jamaica, 1993.
7. Petersen RB, Richardson SL, Chen SG, Parchi P, Urig CB, Gambetti P. The prion protein 178^{Asn} mutation alters processing in a transfected human cell line. *Society for Neuroscience*, Miami Beach, Florida, November 1994.
8. Petersen RB, Tresser NJ, Richardson SL, Gali M, Goren H, Gambetti P. A family with oculoleptomeningeal amyloidosis has a mutation in the transthyretin gene. *American Association of Neuropathologists*, San Antonio Texas, June 1995.
9. Petersen RB. Effect of PrP mutations on PrP metabolism. *Molecular Biology of Prions and Pathology of Prion Diseases*. Banbury Center CSH Laboratory, November 1995.
10. Petersen RB. FFI: genetic and molecular biology findings. *International Workshop on Fatal Familial Insomnia (FFI)*. Bologna, Italy, May 1996.
11. Petersen RB. The inherited prion diseases and the effects of PrP mutations on the metabolism of PrP. *2nd Annual German American Frontiers of Science Symposium sponsored by the German American Academic Council in conjunction with the U.S. National Academy of Sciences, the Alexander Von Humboldt Foundation, the Max Planck Society and the German Academic Exchange*, June 1996
12. Petersen RB, Capellari S, Chen SG, Parchi P, Singh N, Zanusso G, Gambetti P. Effect of Pathogenic Mutations on Processing of the Prion Protein in Transfected Cells: Implications for Pathogenesis. *Neurodegenerative Disorders: Common Molecular Mechanisms*. Ocho Rios, Jamaica, 1997.
13. Petersen RB, Smith MA. Effect of Chronic Expression of Heme Oxygenase-1 in Neuronal Cells: Relevance to Neurodegenerative Diseases. *American Association of Neuropathologists*, Pittsburgh, Pennsylvania, June 1997.

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14. Capellari S, Petersen RB. Studies of the post-translational modifications to the prion protein. *Neurodegenerative Disorders: Common Molecular Mechanisms*, Ocho Rios, Jamaica, 1998.
15. Petersen RB (lecturer). Workshop on "Prions and Aggregating Proteins." Atlanta, GA, June 1998.
16. Petersen RB (lecturer). Prions: From Protein to Pathogen. American Society of Clinical Pathologists Teleconference, June 1998.
17. Curagen Corp, New Haven, CT. August 1998.
18. Institute of Human Virology, University of Maryland. Baltimore, MD. November 1998.
19. Animal Resource Center, Case Western Reserve University, Continuing staff training. November 20, 1998.
20. Prion Disease: Current Health Issues. St. Joseph's Hospital, Milwaukee, WI. May 1999.
21. Prions: From Protein to Pathogen. Milwaukee Academy of Medicine. Milwaukee, WI. May 1999.
22. New Generation- New Approach/ Germany and the USA in an Age of Global History. Sponsored by the German -American Academic Council Foundation. Chicago, IL. November 1999.
23. Prion disease: an Update. Clinical Pathology Conference Series, CWRU, October, 2000:
24. Prion Disease: From Protein to Pathogen. Indiana University of Pennsylvania. Indiana, PA February 9, 2001.
25. Ruminations on the Origins and Impact of Mad Cow Disease. CWRU Women's Alumni Association, September 26, 2001.
26. Ruminations on the Biology of Prion Disease. Department of Neuroscience, CWRU, Cleveland, OH. January 16, 2002.
27. Diagnostics for TSEs: A Case for Simplicity, Keystone Symposium, Breckenridge, CO. April 5, 2003.
28. Prion Safety Training, Pathology P3 Staff, Institute of Pathology, Cleveland, OH. April 15, 2003.
29. Diagnostics for TSEs: A Case for Simplicity, University of Wyoming, Laramie, WY. April 18, 2003.
30. Cell Models of Inherited Human Prion Disease, Veterinary Laboratory Agency-Weybridge, New Haw, Addlestone, Surrey, United Kingdom. August 19, 2003.
31. Ethics of Using Animals in Research, Department of Pathology, CWRU, Sept. 12, 2003.

Publications:

Robert B. Petersen

1. Petersen RB, Hensel CH, Hackett PB. Identification of a ribosome-binding site for a leader peptide encoded by Rous sarcoma virus RNA. *J Virol* **51**: 722-729, 1984.
2. Petersen RB, Hackett PB. Characterization of ribosome binding on Rous sarcoma virus RNA *in vitro*. *J Virol* **56**: 683-690, 1985.
3. Hackett PB, Petersen RB, Albericio F, Gunderson SI, Hensel CH, Palmenberg AC, Barany G. Synthesis *in vitro* of a seven amino peptide encoded in the lead RNA of Rous sarcoma virus. *J Mol Biol* **190**: 45-57, 1986.
4. Petersen R, Lindquist S. The *Drosophila hsp70* message is rapidly degraded at normal temperature and stabilized by heat shock. *Gene* **72**: 161-168, 1988.
5. Hensel CH, Petersen RB, Hackett PR. Effects of alterations in the leader sequence of Rous sarcoma virus RNA on initiation of translation. *J Virol* **63**: 4986-4990, 1989.
6. Petersen RB, Moustakas A, Hackett PB. A mutation in the short 5'-proximal open reading frame on Rous sarcoma virus alters virus production. *J Virol* **63**: 4787-4796, 1989.
7. Petersen RB, Lindquist S. Regulation of HSP70 synthesis by messenger RNA degradation. *Cell Regulation* **1**: 135-149, 1989.
8. Yost HJ, Petersen RB, Lindquist S. RNA metabolism: strategies for regulation in the heat shock response. *Trends In Genetics* **6**: 233-227, 1990.
9. Lindquist S, Petersen RB. Selective translation and degradation of heat shock messenger RNAs in *Drosophila*. *Enzyme* **44**: 147-166, 1990.
10. Hackett RB, Dalton MW, Johnson DP, Petersen RB. Phylogenetic and physical analysis of the 5'leader RNA sequences of Avian retroviruses. *Nuc Acids Res* **19**: 6929-6934, 1991.
11. Petersen RB, Tabaton M, Berg L, Schrank B, Torack RM, Julien J, Vital C, Deleplanque B, Pendlebury WW, Drachman D, Smith TW, Davies P, Martin JJ, Oda M, Montagna P, Autilio-Gambetti L, Lugaresi E, Gambetti P. Analysis of the prion gene in thalamic dementia. *Neurology* **42**: 1859-1863, 1992.
12. Goldfarb LG, Petersen RB, Tabaton M, Brown P, LeBlanc AC, Montagna P, Cortelli P, Julien J, Vital C, Pendlebury WW, Haltia M, Willis PR, Hauw JJ, McKeever PE, Monari L, B Schrank, Swergold GD, Autilio-Gambetti L, Gajdusek C, Lugaresi E, Gambetti P. Fatal familial insomnia and familial creutzfeldt jakob disease: disease phenotype determined by a DNA polymorphism. *Science* **258**: 806-808, 1992.
13. Grant MP, Cohen M, Petersen R, Halmagyi GM, McDougall A, Tusa RJ, Leigh RJ. Abnormal eye movements in Creutzfeldt-Jakob disease. *Ann Neurol* **34**: 192-187, 1993.
14. Monari L, Chen SG, Brown P, Parchi P, Petersen RB, Mikol J, Gray F, Cortelli P, Montagna P, Ghetti B, Goldfarb LG, Gajdusek DC, Lugaresi E, Gambetti P, Autilio-Gambetti L. Fatal Familial Insomnia and Familial Creutzfeldt-Jakob disease: Different prion proteins determined by a DNA polymorphism. *Proc Natl Acad Sci USA* **91**: 2839-2842, 1994.

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15. Smith MA, Kutty RK, Richey PL, Chader GJ, Wiggert B, Petersen RB, Perry G. Heme Oxygenase-1 is associated with the neurofibrillary pathology of Alzheimer Disease. *Am J Pathol* **145**: 42-47, 1994.
16. Yu J, Nagarajan S, Ueda E, Knez JJ, Petersen RB, Medoff ME. Characterization of alternatively spliced PIG-A transcripts in normal and paroxysmal nocturnal hemoglobinuria cells. *Brazilian J Med Biol Res* **27**: 195-201, 1994.
17. Dellavalle RP, Petersen RB, Lindquist S. Preferential deadenylation of Hsp70 mRNA plays a key role in regulating Hsp70 expression in *Drosophila melanogaster*. *Mol Cel Biol* **14**: 3646-3659, 1994.
18. Petersen RB, Tabaton M, Chen SG, Monari L, Richardson SL, Manetto V, Lanska DJ, Markesberry WR, Currier RD, Autilio-Gambetti, L, Gambetti P. Familial progressive subcortical gliosis: presence of prions and linkage to chromosome 17. *Neurology* **45**: 1062-1067, 1995.
19. Parchi P, Castellani R, Cortelli P, Montagna P, Chen SG, Petersen RB, Lugaresi E, Autilio-Gambetti L, Gambetti P. Regional distribution of protease resistant prion protein in Fatal Familial Insomnia. *Ann Neurol* **38**: 21-29, 1995.
20. Premkumar DRD, Smith MA, Richey PL, Petersen RB, Castellani R, Kutty RK, Wiggert B, Perry G, Kalaria RN. Induction of heme oxygenase-1 mRNA and protein in neocortex and cerebral vessels in Alzheimer's disease. *J Neurochem* **65**:1399-1402, 1995.
21. Tabaton M, Rolleri M, Masturzo P, Cammarata S, Angelini G, Hansen LA, Saitoh T, Petersen RB, Perry G, Richey P, Gambetti P, Bertolini S. Apolipoprotein E ε4 allele frequency is not increased in progressive supranuclear palsy. *Neurology* **45**: 1764-1765, 1995.
22. Petersen RB, Tresser NJ, Richardson SL, Gali M, Goren H, Gambetti P. A family with oculoleptomeningeal amyloidosis and dementia has a mutation in the transthyretin gene. *J Neuropathol Exp Neurol* **54**: 413, 1995.
22. Sforza E, Montagna P, Tinuper P, Cortelli P, Avoni P, Ferrillo F, Petersen R, Gambetti P, Lugaresi E. Sleep-wake cycle abnormalities in fatal familial insomnia. Evidence of the role of the thalamus in sleep regulation. *Electroencephalogr Clin Neurophysiol* **94**: 398-405, 1995.
23. Petersen RB, Parchi P, Richardson SL, Urig CB, Gambetti P. Effect of the D178N mutation and the codon 129 polymorphism on the metabolism of the prion protein. *J Biol Chem* **271**: 12661-12668, 1996.
24. Parchi P, Castellani R, Capellari S, Ghetti B, Young K, Chen SG, Farlow M, Dickson DW Sima AAF, Trojanowski JQ, Petersen RB, Gambetti P. Molecular basis of phenotypic variability in sporadic Creutzfeldt-Jakob disease. *Ann Neurol* **39**: 767-778, 1996.
25. Petersen RB, Goren H, Cohen M, Richardson SL, Tresser N, Lynn A, Gali M, Estes M, Gambetti, P. Transthyretin amyloidosis: A new mutation associated with dementia. *Ann Neurol* **41**(3): 307-313, 1997.
26. Parchi P, Capellari S, Chen SG, Petersen RB, Gambetti P, Kopp N, Brown P, Kitamoto T, Tateishi J, Giese A, Kretschmar H. Typing prion isoforms. *Nature* **386**: 232-233, 1997.

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27. Capellari S, Vital C, Parchi P, Petersen RB, Ferrer X, Jarnier D, Pegoraro E, Gambetti P, Julien J. Familial prion disease with a 144bp insertion in the prion protein gene in a Basque family. *Neurology* **49**: 131-141, 1997.
28. Raymond GJ, Hope J, Kocisko DA, Priola SA, Raymond LD, Bossers A, Ironside J, Will RG, Chen S, Petersen RB, Gambetti P, Rubenstein R, Smits MA, Lansbury PT, Caughey B. Molecular Assessment of the transmissibilities of BSE and scrapie to humans. *Nature* **388**: 285288, 1997.
29. Golic MM, Rong Y, Petersen R, Lindquist SL, Golic KG. FLP-mediated DNA mobilization to specific target sites in *Drosophila* chromosomes. *Nuc Acids Res* **25**: 3665-3671, 1997.
30. Singh N, Zanusso G, Chen SG, Fujioka H, Richardson S, Gambetti P, Petersen RB. A cell model of inherited prion disease: prion protein aggregation reverted by low temperature. *J Cell Biol* **272**: 28461-28470, 1997.
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